The changes in the peripheral nerve were those of parenchymatous atrophy, many of the nerve bundles having entirely disappeared, the larger ones showing a loss of contour and great shrinkage. These changes were most marked in the sciatic nerves. Portions of the muscles taken from the flexors and extensors all showed atrophy of the fibres with granular disintegration and infiltration of the areolar tissue with fat, the greater part of the muscle being composed of densely nucleated connective tissue. In the cauda equina the degeneration in the nerve fibres was very extensive, and the pia mater about the cord was thickened and very dense.

The author thinks the patient first suffered from an acute poliomyelitis which was only partially recovered from, and that the crushing of the hand acted as an exciting cause in the development of the disease in the upper extremities, and this, with the multiple neuritis, which was subsequently developed would account for the symptoms. It is also the opinion of the author that the peripheral neuritis was secondary to the cornual changes, as the atrophy of the limbs proceeded indiscriminately and independently of the sensory disturbances. As regards what part the changes in the posterior columns played in the development of the symptoms, he considers it impossible to say, on account of the date of the beginning of the anæsthesia, or whether it was preceded by hyperæsthesia not having been established. The writer does not try to account for the changes in the cortex, or the influence they had in the course or development of the symptoms, but it is to be noted that they were confined to the motor areas.

A CASE OF PROGRESSIVE MUSCULAR ATROPHY, WITH DISEASE OF THE NUCLEI OF THE BULB AND THE CORTEX.

Dr. A. Alzheimer (Archiv. f. Psych., vol. xxiii, part ii, p. 459). The case reported is that of a merchant, 33 years of age, single and descended from a family without neuropathic history. In 1876 the patient suffered from an attack of typhoid fever; in 1877-78 an abscess in the femur troubled him, which resulted in a fistula, continuing till the time of his death. In 1879 there was first noticed a wasting of the thenar eminence. He became infected with syphilis in 1881, and was treated with mercury and no secondary symptoms were manifest. Toward Christmas of 1889 he suffered severely from pain in the stomach and vomiting, and in February, 1890, the acute psychical disturbance presented itself with delirium. When he entered the asylum the muscles of both arms and shoulders showed marked atrophy and fibrillary contractions. No disturbance of sensation or spasm, but an increase of the tendon reflexes. Severe delirium, followed by death, apparently from paralysis of the brain. At the post-mortem was found thickening and cloudiness of the pia mater, atheromatous state of the blood vessels, proliferation of ependyma in the ventricles, dark gray discoloration of the bottom of the fourth ventricle, atrophy of the anterior cornua of the spinal cord, the anterior roots and trunks of the brachial plexus, atrophy of the muscles of the upper extremity, the shoulder girdle and diaphragm, without a transformation of fat, carcinoma of the liver, tumor of the spleen and induration of the lymphatic glands.

The microscopical examination demonstrated simple atrophy of the ganglionic cells in the anterior horns; moderate sclerosis of the anterior ground bundle; in the atrophied muscles many hypertrophied fibres were found; extreme degenerative change in the vessels at the bottom of the fourth ventricle; degeneration in the nuclei of the funiculus gracilis and funiculus cuneatus and of the vagus and auditory nerves, and also in the ganglionic cells of the motorial area, the pulvinar and the corpus striatum; extensive changes in the ganglionic cells of the cortex cerebri. Weight of the brain and membrane, 1330 grm.; cerebrum, 970 grm.; cerebellum, 165 grm.; mid-brain, 165 grm. These last without the membranes. The spinal cord was cut in pieces 1 1-2 ctm. long and some hardened in alcohol, and afterwards stained by Nissl's method for examinations of the cells; others hardened in bichromate solution and stained by Weigert's method for medullated fibres.

In considering the clinical side of the case the author thinks it was impossible to have made a diagnosis of progressive spinal muscular atrophy during the life of the patient; but were it not for increased tendon reflexes it would be more clearly amyotrophic lateral sclerosis. To explain this abnormal myotatic irritability no changes were found in the spinal cord that could be held accountable for it, and the author thinks it is possible that the disease of the ganglia cells in the motorial areas is where the explanation of the problem should be sought for. The author sums up his results in the case as follows:

1. A case of spinal progressive muscular atrophy which agrees clinically and anatomically with the disease as ordinarily described, and which possibly may have been

influenced in its development by the overtraining of the patient in being a *devotee* to rowing and other sports.

2. Degeneration of the nuclei situated beneath the floor of the fourth ventricle. The localization of these pathological changes shuts out the connection of systemic degeneration of the cord, and possibly they were due to the syphilitic infection, and following the degeneration in the basal blood vessels.

3. An extensive disease of the ganglion cells of the cortex cerebri, without any changes worth remarking in the vessels of the cortex, which one must consider as the foundation for the serious psychical disturbance that was manifested. The anatomical examinations gave no apparent interdependence between the before-mentioned pathological changes. The observed changes in the cortex do not agree with those given in other cases of cerebral paralysis. The author does not attempt to give the *raison d'être* of the changes in the internal organs, as the liver and spleen; but the histological changes were minutely and critically worked over. The changes in the ganglion cells, the nerve trunks and muscle fibres are elucidated by carefully prepared plates.

CONCERNING THE PATHOLOGICAL ANATOMY OF CHOREA.

Dr. Kroemer (Archiv. f. Psych. u. Nervenkrank., vol. xxiii, pp. 538-557). After first giving a thorough historical retrospect of the different opinions and theories which have been held concerning the anatomical changes necessary to the production of chorea, the author recites a case which came under his observation in the latter part of her illness and on whom an autopsy was made.

The patient was a thirty-five year old female who had presented no inherited or acquired neuropathic manifestations until her twenty-fourth year, when she remarked a disposition of the head to move involuntarily from one side to the other. Gradually the hands and feet became affected, so that she was almost unable to continue her work as a house-maid. After four years the movements becoming much worse, she was removed to a charity, where she remained six years, and during this period the contractions were so great as to throw her forcibly backward and forward; she was unable to walk alone and generally helpless. It was in her thirty-fourth year that mental aberration was first noticed. Formerly a quiet and ordinary young woman, she mani-